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=> d his

     (FILE 'HOME' ENTERED AT 18:09:04 ON 18 DEC 2002)

     FILE 'EUROPATFULL, PCTFULL, USPATFULL, USPAT2, WPIDS' ENTERED AT
18:09:21
     ON 18 DEC 2002
L1         81130 S CADMIUM#
L2           154 S L1(S)CANCER
L3            47 S L2(S)HUMAN
L4            26 S L3 NOT PY>1999

     FILE 'HOME' ENTERED AT 18:40:14 ON 18 DEC 2002

     FILE 'TOXCENTER' ENTERED AT 18:42:34 ON 18 DEC 2002
L5         84011 S CADMIUM
L6          3159 S L5(S)HUMAN#
L7            23 S L6(S)ADMINIST?
L8            19 S L7 NOT PY>=1999
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=> d ibib abs kwic 11, 1,3,4,7-8

L8 ANSWER 11 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:69273 TOXCENTER
COPYRIGHT: Copyright 2002 BIOSIS
DOCUMENT NUMBER: BA87:65661
TITLE: ORAL CADMIUM CHLORIDE INTOXICATION IN MICE EFFECTS OF PENICILLAMINE DIMERCAPTOSUCCINIC ACID AND RELATED COMPOUNDS
AUTHOR(S): ANDERSEN O; NIELSEN J B
CORPORATE SOURCE: DEP. ENVIRONMENTAL MED. ODENSE UNIV., J. B. WINSLOWSVEJ 19, DK-5000 ODENSE C, DENMARK.
SOURCE: PHARMACOL TOXICOL, (1988) 63 (5), 386-389
CODEN: PHTOEH.
FILE SEGMENT: BIOSIS
OTHER SOURCE: BIOSIS 1989:131008
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20011116

AB The antidotal efficacies of chelators during acute cadmium intoxication has previously been examined in experiments where both a soluble cadmium salt and the chelator were administered parenterally. In the present study, PA, DMSA and related compounds were studied as oral antidotes during oral CdCl2 intoxication. According to the antagonistic effects noted on mortality, peristaltic toxicity and intestinal cadmium uptake, the relative efficacies of the compounds tested were: DMSA > PAD > DMPS > MSA > PA > NAPA. None of the chelators induced major changes in the organ distribution of absorbed cadmium, in particular no increased cerebral deposition of cadmium. This study indicates that, in oral **cadmium** intoxication in **humans**, orally **administered** DMSA would be likely to offer protection against the local toxicity of **cadmium** in the gastrointestinal tract as well as to reduce the risk of systemic toxicity of absorbed **cadmium**.

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L8 ANSWER 1 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:449997 TOXCENTER
DOCUMENT NUMBER: EMIC-106877
TITLE: Effect of 3-aminotriazole on anchorage independence and mutagenicity in cadmium- and lead-treated diploid human fibroblasts.
AUTHOR(S): Hwua Y S; Yang J L
CORPORATE SOURCE: Department of Life Sciences, National Tsing Hua University, Taiwan, Republic of China.
SOURCE: Carcinogenesis, (1998 May) 19 (5) 881-8.
Journal Code: C9T. ISSN: 0143-3334.
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: EMIC
OTHER SOURCE: EMIC MED-98297801
LANGUAGE: English
ENTRY DATE: Entered STN: 20021200
Last Updated on STN: 20021200

AB Cadmium and lead have been shown to induce cellular transformations and gene mutations in cultured rodent cells, as well as tumours in live animals. However, the mechanisms by which these metals cause cellular transformations and mutations in human cells have not been explored. In this study, we investigated the abilities of cadmium and lead to induce anchorage-independent transformations and hprt gene mutations in diploid human fibroblasts. Human fibroblasts were exposed to either cadmium acetate (0-60 microM) or lead acetate (0-2 mM) for 24 h. After removal of the metals, the cells were kept in exponential growth for 7 and 9 days before mutation and anchorage-independence assays were taken, respectively. Both cadmium and lead significantly induced anchorage-independent colonies in dose-dependent manners; the frequencies of anchorage-independent colonies induced by these metals were similar to those induced by N-methyl-N'-nitro-N-nitrosoguanidine at approximately equal cytotoxic dose ranges (30-10% survival). 3-Aminotriazole at non-cytotoxic dosages decreased catalase activity by > 80%, and markedly enhanced cadmium-induced cytotoxicity and anchorage-independent colonies. **Cadmium** uptake by **human** fibroblasts was not affected by 3-aminotriazole co-**administered** with 10 microM of **cadmium**; whereas **cadmium** uptake and accumulation were enhanced 1.5-fold by 3-aminotriazole co-**administered** with 1-2.5 microM of **cadmium**. Lead-induced anchorage-independence or cytotoxicity was not affected by 3-aminotriazole co-treatment; however, 3-aminotriazole did significantly enhance lead uptake and accumulation in human fibroblasts. Neither cadmium- nor lead-induced 6-thioguanine-resistant mutation frequency in human fibroblasts. Co-administering these metals with 3-aminotriazole did not enhance mutations in human fibroblasts. These results suggest that cadmium and lead may both act as tumour promoters in diploid human fibroblasts, and that reactive oxygen species is more important in cadmium- than lead-induced cytotoxicity and anchorage-independence.

AB. . . (30-10% survival). 3-Aminotriazole at non-cytotoxic dosages decreased catalase activity by > 80%, and markedly enhanced cadmium-induced cytotoxicity and anchorage-independent colonies. **Cadmium** uptake by **human** fibroblasts was not affected by 3-aminotriazole co-**administered** with 10 microM of **cadmium**; whereas **cadmium** uptake and accumulation were enhanced 1.5-fold by 3-aminotriazole co-**administered** with 1-2.5 microM of **cadmium**. Lead-induced anchorage-independence or cytotoxicity was not affected by 3-aminotriazole co-treatment; however, 3-aminotriazole did significantly enhance lead uptake and accumulation in.

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L8 ANSWER 3 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:146653 TOXCENTER
COPYRIGHT: Copyright 2002 ACS
DOCUMENT NUMBER: CA12908091667U
TITLE: Effect of 3-aminotriazole on anchorage independence and mutagenicity in cadmium- and lead-treated diploid human fibroblasts
AUTHOR(S): Hwua, Yi-Shi; Yang, Jia-Ling
CORPORATE SOURCE: Molecular Carcinogenesis Laboratory, Department of Life Sciences, National Tsing Hua University, Hsinchu, 300, Taiwan.
SOURCE: Carcinogenesis, (1998) Vol. 19, No. 5, pp. 881-888. CODEN: CRNGDP. ISSN: 0143-3334.
COUNTRY: TAIWAN, PROVINCE OF CHINA

DOCUMENT TYPE: Journal
FILE SEGMENT: CAPLUS
OTHER SOURCE: CAPLUS 1998:345731
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20020521

AB Cadmium and lead have been shown to induce cellular transformations and gene mutations in cultured rodent cells, as well as tumors in live animals. However, the mechanisms by which these metals cause cellular trans- formations and mutations in human cells have not been explored. In this study, the authors investigated the abilities of cadmium and lead to induce anchorage-independent trans- formations and hprt gene mutations in diploid human fibroblasts. Human fibroblasts were exposed to either cadmium acetate (0-60 .mu.M) or lead acetate (0-2 mM) for 24 h. After removal of the metals, the cells were kept in exponential growth for 7 and 9 days before mutation and anchorage-independence assays were taken, resp. Both cadmium and lead significantly induced anchorage-independent colonies in dose-dependent manners; the frequencies of anchorage-independent colonies induced by these metals were similar to those induced by N-methyl-N'-nitro-N-nitrosoguanidine at approx. equal cytotoxic dose ranges (30-10% survival). 3-Aminotriazole at non-cytotoxic dosages decreased catalase activity by >80%, and markedly enhanced cadmium-induced cytotoxicity and anchorage-independent colonies. **Cadmium** uptake by **human** fibroblasts was not affected by 3-aminotriazole co-**administered** with 10 .mu.M of **cadmium**; whereas **cadmium** uptake and accumulation were enhanced 1.5-fold by 3-aminotriazole co-**administered** with 1-2.5 .mu.M of **cadmium**. Lead-induced anchorage-independence or cytotoxicity was not affected by 3-aminotriazole co-treatment; however, 3-aminotriazole did significantly enhance lead uptake and accumulation in human fibroblasts. Neither cadmium-nor lead-induced 6-thioguanine-resistant mutation frequency in human fibroblasts. Co-administering these metals with 3-aminotriazole did not enhance mutations in human fibroblasts. These results suggest that cadmium and lead may both act as tumor promoters in diploid human fibroblasts, and that reactive oxygen species is more important in cadmium than lead-induced cytotoxicity and anchorage-independence.

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L8 ANSWER 4 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:118152 TOXCENTER
COPYRIGHT: Copyright 2002 ACS
DOCUMENT NUMBER: CA12822267093Z

TITLE: Sister chromatid exchanges in human lymphocytes treated in vitro with cadmium in Go and S phase of their cell cycles
AUTHOR(S): Saplakoglu, Umay; Iscan, Mesude
CORPORATE SOURCE: Middle East Technical University, Department of Biology, Ankara, 06531, Turk..
SOURCE: Mutation Research, (1998) Vol. 412, No. 2, pp. 109-114. CODEN: MUREAV. ISSN: 0027-5107.
COUNTRY: TURKEY
DOCUMENT TYPE: Journal
FILE SEGMENT: CAPLUS
OTHER SOURCE: CAPLUS 1998:137244
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20020605

AB Sister chromatid exchanges (SCEs) were analyzed in human phytohemagglutinin-activated peripheral lymphocyte cultures exposed to varying concns. (10-7-10-3 M) of cadmium chloride in vitro at two different stages of the cell cycle, Go and early S phase. When **cadmium** chloride was **administered** at the Go phase, no increase in the SCEs were obsd. for the doses 10-6 and 10-5 M; concns. equal to or larger than 10-4 M **cadmium** chloride were lethal to **human** lymphocytes in our exptl. conditions. A highly statistically significant increase was obsd. in the SCE frequency with increasing cadmium chloride concn. (10-7-10-4) when cadmium was administered at the early S phase, which was 24 h after culture initiation. The increase in SCE frequency was higher when the cultures were terminated at 54 h, compared to termination at 72 h. In order to examine the effects of cadmium administered at the S phase on SCE frequency in different individuals, 10-5 M concn. was used and the cultures were terminated at 54 h after culture initiation. A 2- to 3-fold increase in the SCE frequency was obsd. in all six individuals examd. A progressive decrease in the proliferative index was also obsd. by increasing cadmium chloride concn. These results demonstrate that the genotoxicity of cadmium chloride may be changed depending on the stage of the cell cycle in human lymphocytes. This may be one of the reasons of contradictory findings in the literature.

AB. . . M) of cadmium chloride in vitro at two different stages of the cell cycle, Go and early S phase. When **cadmium** chloride was **administered** at the Go phase, no increase in the SCEs were obsd. for the doses 10-6 and 10-5 M; concns. equal to or larger than 10-4 M **cadmium** chloride were lethal to **human** lymphocytes in our exptl. conditions. A highly statistically significant increase was obsd. in the SCE frequency with increasing cadmium chloride. . .

L8 ANSWER 7 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:127246 TOXCENTER
COPYRIGHT: Copyright 2002 ACS
DOCUMENT NUMBER: CA12611140713E
TITLE: Heat-shock and cadmium chloride increase the vimentin mRNA and protein levels in U-937 human promonocytic cells
AUTHOR(S): Vilaboa, Nuria E.; Garcia-Bermejo, Laura; Perez, Concepcion; De Blas, Elena; Calle, Consuelo; Aller, Atricio
CORPORATE SOURCE: Centro de Investigaciones Biologicas, CSIC, Madrid, 28006, Spain.
SOURCE: Journal of Cell Science, (1997) Vol. 110, No. 2, pp.

201-207.
CODEN: JNCSAI. ISSN: 0021-9533.
COUNTRY: SPAIN
DOCUMENT TYPE: Journal
FILE SEGMENT: CAPLUS
OTHER SOURCE: CAPLUS 1997:123282
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20020626

AB Heat-shock for 2 h at 42.degree.C, or the **administration** for 3 h of 100 or 150 .mu.M **cadmium** chloride, inhibited the subsequent proliferation activity, induced the expression of functional differentiation markers, and caused an increase in the amt. of the stress-responsive HSP70 protein in U-937 **human** promonocytic cells. In addn., both heat and cadmium produced an increase in the amt. of the intermediate filament protein vimentin, as detd. by immunoblot and immunofluorescence assays. By contrast, the amts. of actin and .beta.-tubulin were not significantly altered. The amt. of vimentin mRNA was also increased during recovery from stress, indicating that vimentin expression was not exclusively regulated at the protein level. Although cadmium caused an early, transient stimulation of c-jun and c-fos expression and AP-1 binding activity, heat-shock failed to alter both protooncogene expression and transcription factor binding, indicating that the stress-induced vimentin increase was not the result of AP-1-mediated transcriptional activation. Finally, it was obsd. that the rate of decay of vimentin mRNA upon actinomycin D administration was decreased in heat- and cadmium-pretreated cells in comparison to untreated cells. These results indicate that stress treatments cause an increase in vimentin levels in promonocytic cells, which may be explained at least in part by transcript stabilization.

AB Heat-shock for 2 h at 42.degree.C, or the **administration** for 3 h of 100 or 150 .mu.M **cadmium** chloride, inhibited the subsequent proliferation activity, induced the expression of functional differentiation markers, and caused an increase in the amt. of the stress-responsive HSP70 protein in U-937 **human** promonocytic cells. In addn., both heat and cadmium produced an increase in the amt. of the intermediate filament protein vimentin,. . .

L8 ANSWER 8 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:138179 TOXCENTER
COPYRIGHT: Copyright 2002 ACS
DOCUMENT NUMBER: CA12213150991A
TITLE: Effect of cadmium on human ovarian cancer cells with acquired cisplatin resistance
AUTHOR(S): Lee, Kang Bo; Parker, Ricardo J.; Reed, Eddie
CORPORATE SOURCE: Clinical Pharmacology Branch, National Cancer Institute, Building 10, Room 12C103, Bethesda, MD, 20892, USA.
SOURCE: Cancer Letters (Shannon, Ireland), (1995) Vol. 88, No. 1, pp. 57-66.
CODEN: CALEDQ. ISSN: 0304-3835.
COUNTRY: UNITED STATES
DOCUMENT TYPE: Journal
FILE SEGMENT: CAPLUS
OTHER SOURCE: CAPLUS 1995:320406
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20020903

AB Cadmium (Cd) dichloride is a compd. that has teratogenic, mutagenic, and carcinogenic properties. Recent reports have suggested the possibility

that this compd. may also have tumor suppressive properties in some settings. For these reasons, we have studied the subcellular pharmacol. profile of elemental **cadmium** in **human** ovarian cancer cells, when **administered** as **cadmium** dichloride. The cell lines A2780 and A2780/CP70 were used, which are well characterized with respect to their cellular response to platinum-based compds. Cd was measured in all expts. with the use of at. absorbance spectrometry with Zeeman background correction. In both cell lines, there were direct relationships between; drug dose and cellular accumulation of drug; cellular accumulation of drug and DNA damage levels; and DNA damage levels and cytotoxicity. These cell lines differed in that the cisplatin-resistant A2780/CP70 cell line, was also comparatively resistant to cadmium dichloride. This enhanced cellular resistance appeared to be mediated through decreased drug accumulation, and increased cellular tolerance to higher levels of DNA damage. Total genomic DNA repair and cytosolic inactivation of drug appeared not to differ substantively between these two cell lines.

AB. . . also have tumor suppressive properties in some settings. For these reasons, we have studied the subcellular pharmacol. profile of elemental **cadmium** in **human** ovarian cancer cells, when **administered** as **cadmium** dichloride. The cell lines A2780 and A2780/CP70 were used, which are well characterized with respect to their cellular response to. . .

L8 ANSWER 11 OF 19 TOXCENTER COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1989:69273 TOXCENTER
COPYRIGHT: Copyright 2002 BIOSIS
DOCUMENT NUMBER: BA87:65661
TITLE: ORAL CADMIUM CHLORIDE INTOXICATION IN MICE EFFECTS OF PENICILLAMINE DIMERCAPTOSUCCINIC ACID AND RELATED COMPOUNDS
AUTHOR(S): ANDERSEN O; NIELSEN J B
CORPORATE SOURCE: DEP. ENVIRONMENTAL MED. ODENSE UNIV., J. B. WINSLOWSVEJ 19, DK-5000 ODENSE C, DENMARK.
SOURCE: PHARMACOL TOXICOL, (1988) 63 (5), 386-389
CODEN: PHTOEH.
FILE SEGMENT: BIOSIS
OTHER SOURCE: BIOSIS 1989:131008
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20011116

AB The antidotal efficacies of chelators during acute cadmium intoxication has previously been examined in experiments where both a soluble cadmium salt and the chelator were administered parenterally. In the present study, PA, DMSA and related compounds were studied as oral antidotes during oral CdCl2 intoxication. According to the antagonistic effects noted on mortality, peristaltic toxicity and intestinal cadmium uptake, the relative efficacies of the compounds tested were: DMSA > PAD > DMPS > MSA > PA > NAPA. None of the chelators induced major changes in the organ distribution of absorbed cadmium, in particular no increased cerebral deposition of cadmium. This study indicates that, in oral **cadmium** intoxication in **humans**, orally **administered** DMSA would be likely to offer protection against the local toxicity of **cadmium** in the gastrointestinal tract as well as to reduce the risk of systemic toxicity of absorbed **cadmium**.

AB. . . the organ distribution of absorbed cadmium, in particular no increased cerebral deposition of cadmium. This study indicates that, in oral **cadmium** intoxication in **humans**, orally **administered** DMSA would be likely to offer protection against the local toxicity of **cadmium** in the gastrointestinal tract as well as to reduce the risk of systemic toxicity of absorbed **cadmium**.

Medico Abstracts
ON
RENAL DISORDERS

Bascands JL,Cabos - Boutot C,Manual Y,Girolami JP

Author	Title
Bascands JL,Cabos-Boutot C,Manual Y,Girolami JP	Pretreatment with low doses of Cadmiun (Cd) protects rat renal mesangial cells against the direct toxic effects of Cadmiun

Document Type	Location	Language
Journal Article	Glasgow Homoeopathic Library	English

Source	Year, Volume & Pages
J OMHI	1990 Dec;3(3): 9-13

Key Terms	RESEARCH
Minor Terms	metals, trace elements, rats, poisoning, prevention, homeopathy
Entry Terms	cadmiun

Abstract

We have compared response of renal mesangial cells to different does of cadmium. The effect of cadmium was assessed by measuring the growth rate of an homogeneous rat renal cell line. The cells grown in the presence of 10-6 MCd exhibited a 37+/- 6% mortality rate within 24 h. When the cells were first placed in the presence of 10-15 or 10-20 MCdCl2 for 24, 48, 72 and 98 hours respectively and then placed in the presence of 10-6 MCdCl2, the toxic effect initially observed with this concentration was significantly reduced. This decrease in Cd cytotoxicity was also dependent on the pretreatment time. The maximum resistance effect was obtained after 72 hours of pretreatment, a 45=/-6% increase in survival rate was observed. However, 96 hours pretreatment did not increase the protective effects. In conclusion renal mesangial cells alone may exhibit a transient protective mechanism against a toxic dose of cadmium itself at doses as low as 10-15 and 10-20 M. The induction of metallothionein is likely to be involved.

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